

In 13 of the affected patients cutaneous blood flow and sweating were recorded during several attacks. Our results of blood flow measurements tally with those of Dr Ginsburg and Ms O'Reilly. Sweating was recorded with an evaporimeter (Ep 1, Servomed, Stockholm) and cutaneous blood flow with a laser Doppler flow meter (Periflux, Perimed, Stockholm). The increase in the rate of evaporation exceeded 60 g/m²/h in 10 attacks, varied from 10 to 60 g/m²/h in five attacks, and was <10 g/m²/h in seven attacks. The cutaneous blood flow increased synchronously with the increase in evaporation. The intensity of the attacks as experienced by the patients corresponded closely with the recorded measurements.

E VARENHORST
T FRÖDIN
G ÅLUND

Departments of Urology and Dermatology,
University Hospital,
S-581 85 Linköping,
Sweden

¹ Huggins C, Stevens RE, Hodges CV. Studies on prostatic cancer. II. The effect of castration on advanced carcinoma of the prostate gland. *Arch Surg* 1941;43:209-23.

however,⁵ and involvement of the myocardium can be fatal.² Fat or bone marrow embolism may be responsible for the rare cases of sudden death in sporting events causing skeletal jarring and soft tissue injury, but special techniques would be required to show this.

Mr Allister's patient developed several focal neurological problems consistent with embolism of the nervous system.⁶

PHILIP B JAMES

Wolfson Institute of Occupational Health,
University of Dundee,
Dundee

¹ Palmovic V, McCarroll JR. Fat embolism in trauma. *Archives of Pathology* 1965;80:630-5.

² Pyun KS, Katzenstein RE. Widespread bone marrow embolism with myocardial involvement. *Archives of Pathology* 1970;89:378-81.

³ Chin-Shen L, Zak FG. Paradoxical bone marrow coronary embolism following cardiopulmonary resuscitation. *JAMA* 1982;248:33.

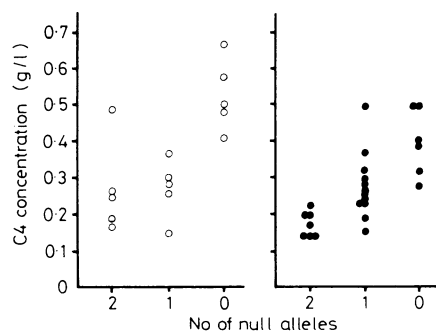
⁴ Roessmann U, Zarchin LE. Cerebral bone marrow embolus after closed chest cardiac massage. *Arch Neurol* 1979;36:58.

⁵ Dehand FH, Bennett WA. Death due to bone-marrow and tumour embolisation in the absence of fracture. *Archives of Pathology* 1957;63:13-6.

⁶ James PB. Evidence for subacute fat embolism as the cause of multiple sclerosis. *Lancet* 1982;i:380-6.

Low C4 concentrations in insulin dependent diabetes mellitus

SIR,—We must agree with the suggestion by Dr D Vergani and others (19 March, p 926) that an inherited deficiency of the fourth component of complement (C4D) is associated with insulin dependent diabetes mellitus. As we and others have shown previously,^{1,2} two of three high risk supratypes (HLA-B8 Bf*S C4A*Q0 C4B*1 DR3 and HLA-B18 Bf*F1 C4A*3 C4B*Q0 DR3) contain null alleles at the C4 loci. Furthermore, we have observed, as has been shown by others,³ that serum C4 concentration is related in general to the number of C4 null alleles (figure).



Serum C4 concentrations versus number of C4 alleles in 15 patients with insulin dependent diabetes mellitus (left) and in 25 healthy subjects (right). The number of null alleles was assigned after C4 allotyping using neuraminidase treated plasma⁴: 2=homozygous deficiency at one C4 locus and two separate alleles present at the other; 1=three separate alleles with one null allele deduced because of C4:B densitometric ratios⁴ of either 2 or 1/2; 0=four separate alleles.

We examined C4 concentrations in 15 patients with insulin dependent diabetes mellitus selected according to whether they had none, one, or two C4 null alleles. Four of five patients with two null alleles had C4 concentrations in the range 0.15-0.26 g/l, whereas all five patients without null alleles had concentrations in the range 0.41-0.67 g/l. The group with one null allele was intermediate.

There can be little doubt that C4 null alleles are at least partly responsible for the relatively low C4 concentrations found by Dr Vergani and colleagues. Other factors may also be implicated (rate of consumption and synthesis, etc) but we see no cause to postulate additional genetic factors on the present evidence. If a search for such factors is undertaken we would urge that allowance be made for the contribution of the C4 loci within the major histocompatibility complex.

ROGER L DAWKINS
GABRIEL UKO
FRANK T CHRISTIANSEN
PETER H KAY

Department of Clinical Immunology,
Royal Perth Hospital,
Perth, Western Australia 6001

¹ Dawkins RL, Christiansen FT, Kay PH, et al. Disease associations with complement, supratypes and haplotypes. *Immunol Rev* 1983;70:5-22.

² Raum D, Awdeh ZL, Alper CA. Bf types and mode of inheritance of insulin dependent diabetes mellitus (IDDM). *Immunogenetics* 1981;12:59-74.

³ Awdeh ZL, Ochs HD, Alper CA. Genetic analysis of C4 deficiency. *J Clin Invest* 1981;67:260-3.

⁴ Christiansen FT, Uko G, Dawkins RL, McCuskey J, Zilko PJ. Complement allotyping in SLE: association with C4A null. In: Dawkins RL, Christiansen FT, Zilko PJ, eds. *Immunogenetics in rheumatology*. Amsterdam: Excerpta Medica, 1982:229-34.

"Locomotor disability in general practice"

SIR,—I was disappointed with Dr Brian McAvoy's critique of *Locomotor Disability in General Practice*. Quite simply he may have missed the point. That he is surprised by finding back pain and sciatica mentioned in connection with gait disturbance seems to me an excellent reason for Dr McAvoy and others like him to read this book. The criticism of it being a minitextbook of orthopaedics cannot be justified. Thirty per cent of general practice is concerned with locomotor disability. That this book concentrates on describing examination and assessment of such disability is a practical contribution to redress the balance between hospital and general practice.

JONATHAN NOBLE

Hope Hospital,
Salford M6 8HD

Screening in general practice

SIR,—I was fascinated by the article by Dr John F Grace (27 August, p 589), but I wonder if the cervical cytology was included to pay for the screening programme? In our practice, of similar size to the screening practice, we have been performing routine cervical cytological examinations for the past eight years, and in the past year with the help of a computerised age-sex register and personal letters asking patients to attend for a smear. Disease found and treated included: malignant ovarian cyst; anaemia due to fibroids; non-consummation of marriage; two carcinomas in situ; many monilial, trichomonal, and actinomycetes infections; two malignant melanomas on the soles of the feet (we use the left lateral position); and one case of acute appendicitis. Not in the screening, but referred from the local family planning clinic for treatment of persistent inflammatory smears (three), was a woman with a fully developed carcinoma of the cervix, which needed only a speculum examination to diagnose it.

Medicine without signs

SIR,—Most GPs have been familiar with psychosomatic illness for a long time, but the question of what to do with it puts the emphasis on the part of Dr Geoffrey Lloyd's paper (20 August, p 539) headed "In search of explanations." For doctors familiar with Michael Balint's *The Doctor, the Patient and his Illness* it is obvious. The gist of it is that the patient is motivated to produce physical symptoms by unconscious underlying problems. The accent is on "unconscious"—that is, non-aware.

"The dual task in diagnosis" therefore remains with the doctor, who has to be alert to the patient's presentation. No "increasing psychological awareness" in patients will change things. Emotional blocking creates a blind spot in the individual patient, and what he can discern in others, he cannot see in himself. It is not only insight and understanding of the underlying problems that are required, but trained help to work them through. This will assure a decrease in functional illnesses. With insight alone the conflicts will persist and will in no time create a new set of problems.

ANNE L ZWEIG

Haslemere,
Surrey GU27 1AA

Cardiac arrest after crush injury

SIR,—Although biochemical abnormalities may be important in crush injuries, Mr Charles Allister (20 August, p 531) has overlooked a documented mechanism that could account for sudden death after such an injury—embolism by bone marrow or fat. Crushing, even in the absence of detectable fracture, can release large quantities of depot fat into the circulation, which, after passage through the pulmonary vasculature, can cause extensive systemic embolism.^{1,2} Coronary³ and cerebral⁴ arterial occlusions by fragments of bone marrow have recently been shown in patients sustaining rib fractures during external cardiac massage. Bone marrow embolism has also been described in the absence of fracture,